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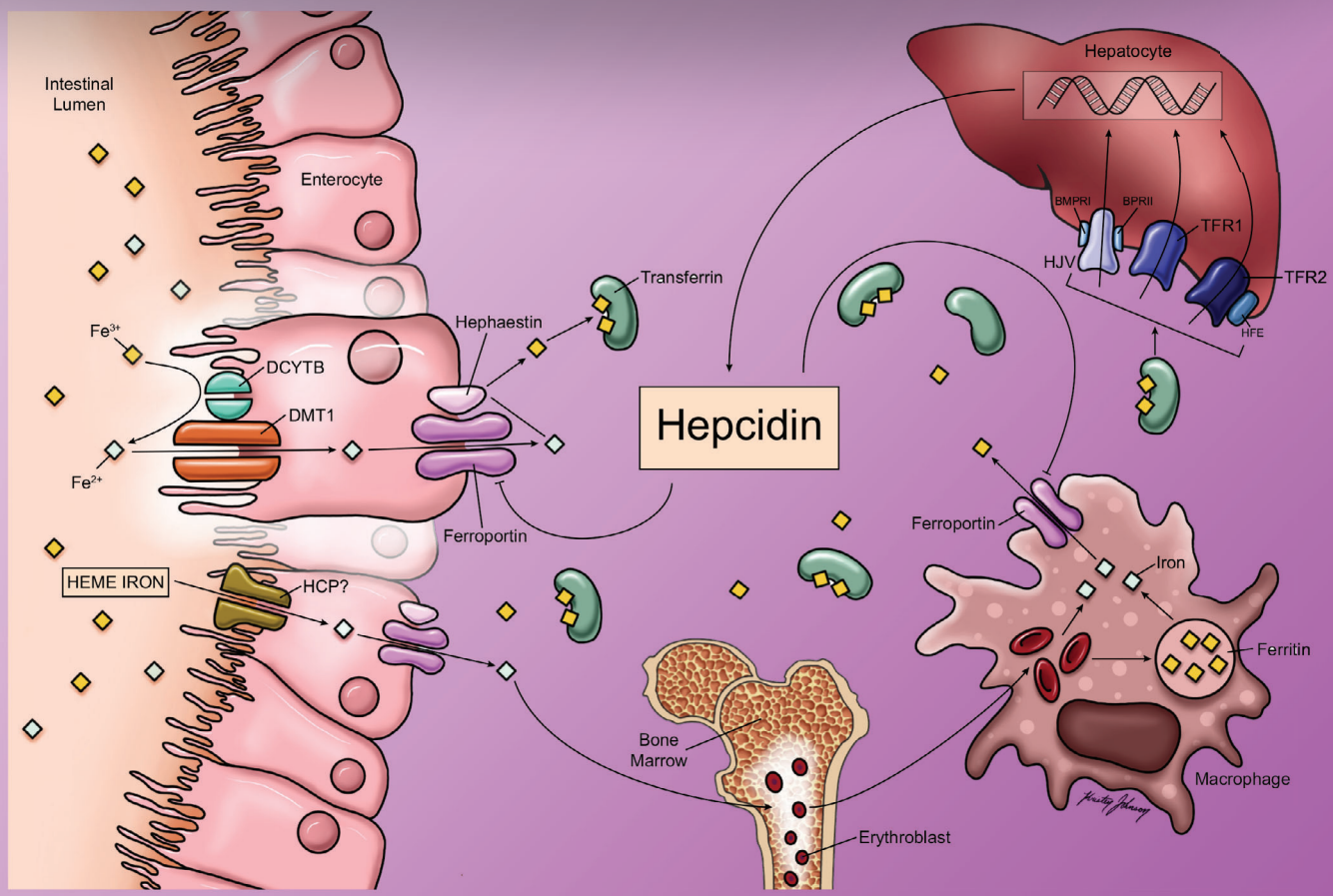

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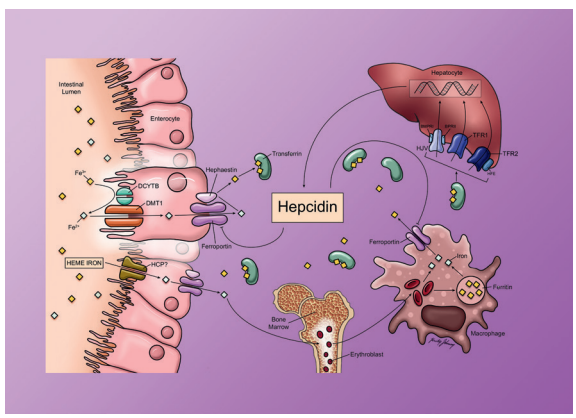
Reviews



Cover Figure: Hepcidin, the master regulator of iron absorption and secretion. Plasma iron levels are controlled primarily at the level of absorption by duodenal enterocytes and RES macrophages. Heme-iron can also be directly absorbed from the gastrointestinal tract through a carrier protein, possibly heme carrier protein. Non-heme (inorganic) iron is absorbed via divalent metallic transporter 1 after conversion from ferric (Fe 3+) to ferrous (Fe 2+) iron through duodenal cytochrome b-related ferric reductase. RES macrophages acquire iron through erythrophagocytosis. Efflux of iron from both enterocytes and RES macrophages occurs through the iron export protein ferroportin. Fe 2+ is reduced back to ferric iron (Fe 3+) through hephaestin before being transported out of the enterocyte. Iron is bound to transferrin as it enters the plasma, which then travels through the circulation where it is taken up by various organs and used in the development of erythrocytes and other biological processes. The principal regulator of iron levels is the hormone hepcidin, which is produced by hepatocytes. As iron stores increase, hepcidin inhibits iron efflux from both duodenum enterocytes and RES macrophages through down-regulation of ferroportin. Serum iron levels influence hepcidin expression through the interaction of the HFE protein with TFR1/TFR2 along with BMP6 and HJV. Abbreviations: DCYTB, duodenal cytochrome b-related ferric reductase; DMT1, divalent metallic transporter 1; HCP, heme carrier protein.

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